INDEX NUMBER

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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

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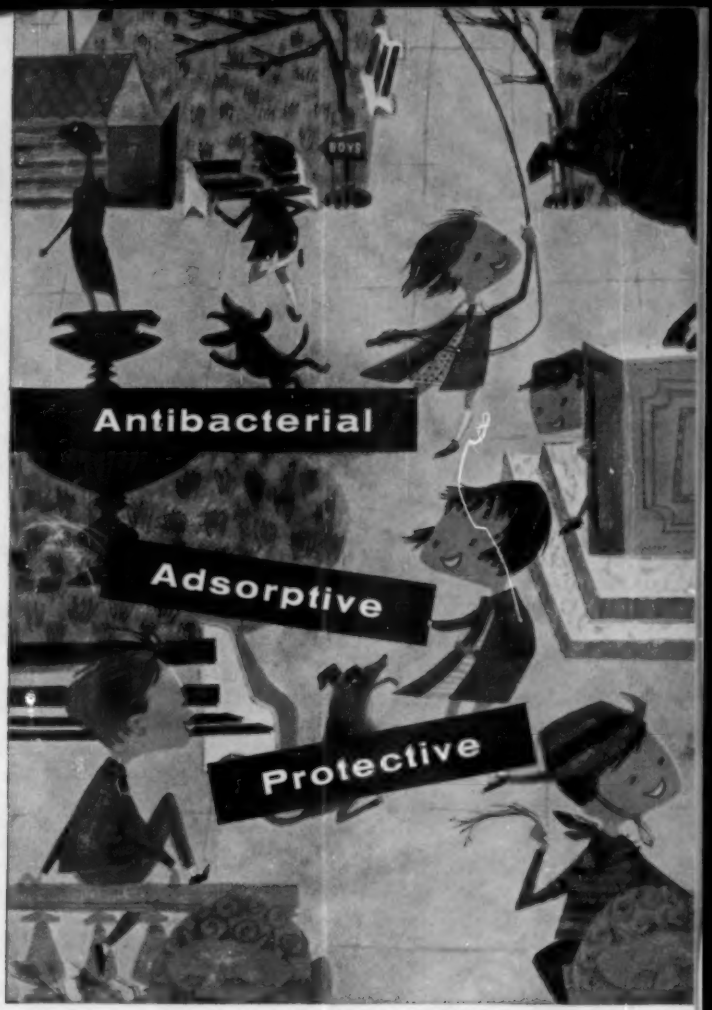

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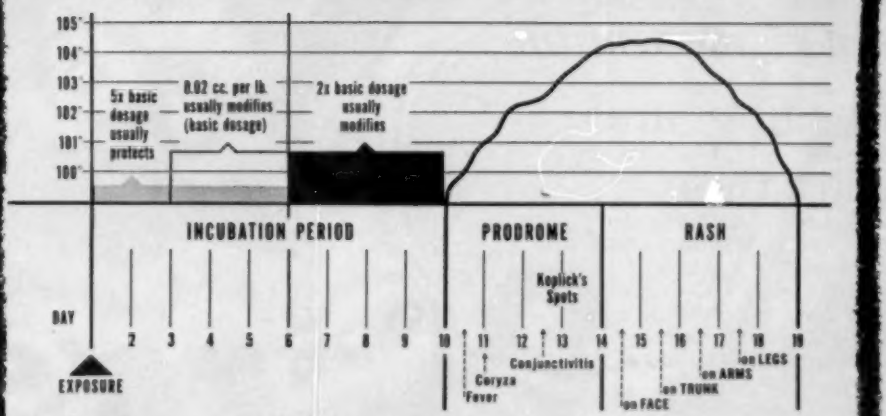

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BIRTH INJURIES OF THE NEWBORN*

MORPHOLOGY, PATHOGENESIS, CLINICAL PATHOLOGY AND PREVENTION OF BIRTH INJURIES OF THE NEWBORN

PH. SCHWARTZ, M.D.**

Warren, Penna.

Many types of intra-uterine lesions — such as infection, intoxication, trauma, disturbances of placentation as well as genic germinal defects and constitutional conditions of the blood, — may cause the child to die in utero or to be born with severe injuries, and then be subject to a miserable life. The birth process itself is rather hazardous: placenta previa, early separation of the placenta, umbilical abnormalities, narcosis, and so forth, which may cause suffocation (anoxia) of the fetus due to loss of blood, or interference with the circulation of the blood and internal respiration. In spite of the importance of such conditions, discussion of them is not included here.

The aims of this paper are: (1) to describe the most important types of *mechanically produced lesions* to which newborn children are exposed during the birth process; (2) to define the most common sequelae found in children and adults which result from such conditions; (3) to point out the most important factors in the pathogenesis of the birth injury; and (4) to indicate the possibilities of prevention of the birth lesion.

*Department of Pathology, Warren State Hospital, Warren, Penna. Department of Welfare, Commonwealth of Pennsylvania.

**Prof. of General Pathology and Pathological Anatomy; Former Director of the Pathological Institute of the University of Istanbul; Prof. of the University of Frankfurt-Main (Germany), on Leave of Absence.

BIRTH IS A BRUTAL PROCESS

Types of mechanically produced lesions of the newborn. Because of the mechanical stress accompanying it, birth is, almost without exception, a brutal process which endangers the life and health of the child. The danger appears to be increased by primiparity, by prematurity; it is accentuated by postmaturity, by malpresentations, and, by an abnormally rigid or narrow birth canal. The danger is less when the child is of normal size, full term, presenting normally, and when the mother is young, well proportioned, and multiparous.

The mechanically produced lesions of the newborn can be divided into two major groups.

1. *Cases in which the results of a common trauma are present.* These include cutaneous contusion; rupture of the dural folds caused by bitemporal or fronto-occipital compression of the cranium; dislocation and overlapping of the cranial bones during the passage through the pelvic outlet; and depression and deformation of the cranial bones by the promontory, and so forth. In this group also belong injuries caused by manual or instrumental obstetrical interference; dislocation and fracture of the extremities and of the vertebral column; or dislocation, depression and fracture of the cranial bones; and rupture of peripheral nerves, etc. Lesions of this group, however, are of secondary importance insofar as they are of frequent occurrence; on the other hand severe injuries of this type are relatively infrequent.

2. *Lesions caused by the direct and indirect effect of the action of the most important driving force of the birth process, the "lower-pressure" or "release"-principle.* These types of lesions are of paramount importance because of their frequency and cerebral localization. They are also of extreme interest from the morphological, clinical and preventive aspects. Accordingly, we will consider first the lower-pressure-principle, and its effects upon the body of the newborn.

BIRTH, A PROCESS OBEYING THE LAWS OF HYDRODYNAMICS

The birth of the child results from an automatic expulsive process. Although associated contractions of the diaphragm and the abdominal musculature are important, the energy required is delivered mainly by the contraction of the uterine muscle.

The uterine muscle contracts intermittently, each contraction

causing a marked diminution of the size of the uterus and a rise of the intra-uterine pressure to 250 mm. Hg. and higher. During relaxation of the uterus, the intra-uterine pressure drops to its normal preparturition level.

To understand the function of the birth mechanism, it is of utmost importance to know that the fetus in utero is suspended in the fluid contained within the amniotic sac. Therefore, during the entire birth process, particularly prior to the rupture of the amnion, the increases in pressure produced by the contraction of the uterus act only indirectly on the fetal body. Thus, *the birth mechanism can be considered as obeying the laws of hydrodynamics.* The force produced by the muscular contraction is transmitted through the medium of the amniotic fluid to the body of the fetus which then moves in the direction of least resistance, namely, towards the os uteri.

THE LOWER-PRESSURE-PRINCIPLE AS THE MAIN DRIVING FORCE OF THE BIRTH PROCESS

If a suction apparatus is applied to any part of the body surface, a swelling develops due to the difference in pressure between the atmosphere and the affected area. The greater the difference in pressure, the larger the swelling. The suction alters the flow of lymph and blood, which is more pronounced, with a greater difference in pressure.

A similar phenomenon may be observed during the expulsion of the fetus from the uterus. The fetus is protected from any change in the distribution of its body fluids, in spite of the rising intra-uterine pressure during labor, as long as the amnion is intact. The conditions suddenly change, however, following the rupture of the amniotic sac. Then, the loss of the amniotic fluid in the protruding portion of the sac allows the presenting part, usually the head, to be forced into the os uteri because it is subjected to a powerful suction due to the relatively lower atmospheric pressure during the uterine contractions. Thus the head closes the os uteri like a ball valve. The average difference in pressure between the uterine cavity and the atmosphere during the period of expulsion can be represented by a water column 3 meters high and 80 cm. in diameter, equivalent to 24 kg. in weight. It is easy to understand that the effect of such a suction force is an accumulation of body fluids at the site of the presenting part. When the presentation is

a breech, deeply penetrating hemorrhages and necroses on the skin and muscles of the buttocks sometimes develop. If the back or face are presenting, similar injuries can be observed in their soft tissues. The most common, however, is the vertex presentation.

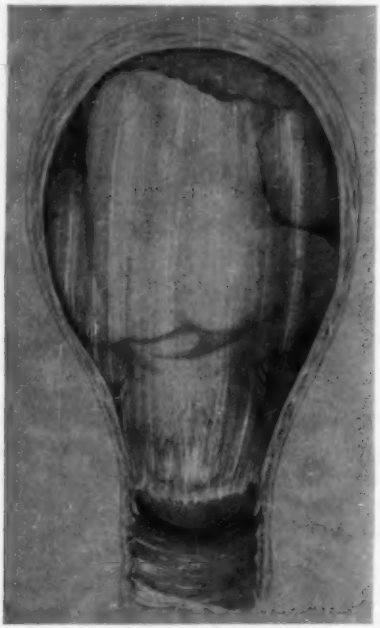


Fig. 1. Intra-uterine position of fetus after the rupture of the amnion bag. The head closes the widely opened os uteri like a ball valve.

Here, the skull and the brain are the organs most frequently injured by the suction effect of the release principle; they are subjected to deformation ("moulding"); in addition, the birth swelling (caput succedaneum), and a cephalhematoma may result in the presenting part; also, pial and cerebral hemorrhages and brain softening may develop.

We have investigated the lower-pressure-principle experimentally. A suction cup was attached to the heads of newborn animals and pressure differences were produced which corresponded to those present in human birth. In this way changes were induced comparable to birth lesions in newborn infants, that is, moulding

of the skull, caput succedaneum, and hemorrhages in the skull and periosteum. It was also possible to produce pial and cerebral hemorrhages.

We do not intend to minimize the common mechanical causes of birth injury mentioned earlier. Nevertheless, we believe that *the importance of the lower-pressure-principle should be particularly emphasized for it is the cause of the most characteristic birth lesions*. Also, recognition of the importance of the release effect in the etiology of birth injuries may become instrumental in their prevention.

CRANIAL BIRTH TRAUMA AND CEREBRAL CIRCULATORY DISTURBANCES

The release effect, the most important mechanical driving force of birth, is doubtless a physiological phenomenon occurring during the period of expulsion. However, it also may be the cause of severe injuries to the fetus. In vertex presentations, when the membranes have ruptured, important sectors of the large cranial venous sinuses, protected only by the thin layer of the scalp and the open sutures and the fontaneles, become exposed to the action of the lower-pressure effect. Thus, it may be presumed that, in many cases, the release effect is capable of producing circulatory disturbances transmitted to deeper parts of the brain. In fact, investigations have shown that almost *every baby born normally suffers some disturbance of the cranial and even the cerebral circulation* as a result of the release effect. This has been established primarily in newborn infants who died during or shortly after birth. However, clinical observation has also shown that many surviving children suffered cerebral circulatory disturbances due to birth trauma.

The longitudinal and straight sinuses converge at the site which is the area most commonly presenting in birth. Therefore, they may be directly affected by the suction force of the lower-pressure-effect. Now, what are the consequences of a forceful suction exerted upon the longitudinal sinus? The blood in the longitudinal sinus normally flows with a slightly negative pressure. The suction effect not only stops this flow, but it also draws the venous blood of the pia and falx into the longitudinal sinus, flooding it and its branches. *Thus, stasis, hemorrhage, and thrombosis develop in the suction area, according to the pressure differences created*

by the uterine contractions. The overfilling of the longitudinal sinus involves the straight and the transverse sinuses too. It is clear, however, that the vein of Galen is more liable to be affected than the sinuses, because the flow of blood from the intracerebral veins through the Galenic vein is impeded by the narrowness and rigidity of the straight sinus, and by the acute angle this sinus forms with the axis of the vein of Galen. The congested blood in the vein of Galen cannot flow freely.

It seems that the vein of Galen, — which is short and strikingly wide, — functions not only as a simple vascular conduit, but also as a container and a regulator of the cerebral venous circulation, capable, if necessary, of accumulating considerable amounts of blood. Its regulatory mechanism plays an important role during birth and when overburdened, often becomes paralyzed.

Fig. 2 shows the branches of the Galenic system: the cerebellar

Fig. 2. The system of the Great vein of Galen. (We injected a blue colored mass into the Galenic vein). (*g*) Great vein of Galen; (*o*) internal occipital vein; (*b*) basal vein of Rosenthal; (*lv*) lateral ventricular vein; (*ci*) internal cerebral vein; (*ch*) chorioidal vein; (*s*) septum pellucidum; (*ca*) caudate nucleus showing branches of the terminal vein on its surface; (*m*) white matter of cerebral hemispheres injected with the blue dye; (*e*) epiphysis.

vein, the internal occipital vein, the basal vein of Rosenthal, the pineal vein, the lateral ventricular vein, the choroidal vein, the vein of the septum pellucidum, the internal cerebral vein, and the terminal vein. *All of these vessels, and their intracerebral ramifica-*

tions, may be involved by the lower-pressure-effect and become the sites of typical birth traumatic circulatory disturbances.

Rough injuries of the Galenic vein occur relatively infrequently. When they are present, the lesion is mostly characterized by thrombotic occlusion, often accompanied by enormous dilatation of the vessel. The internal cerebral vein is seldom involved, but one of its branches, the *terminal vein*, appears to be affected so often that its lesion may be regarded as the most characteristic of all cerebral injuries resulting from birth trauma. There are hemorrhages, which relate to the anterior root of the terminal vein, in-

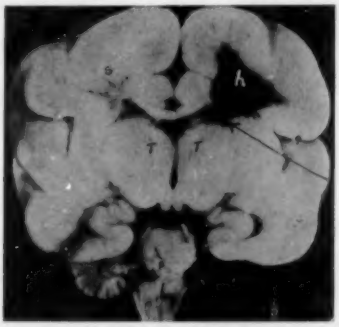


Fig. 3. Birth traumatic hemorrhage in the cerebral white matter. The stem of left terminal vein showed thrombosis and perivascular bleeding. Premature child. Note discoloration of periventricular area of the cerebral white matter on *right* side caused by microscopic hemorrhages and a beginning softening. Arrow indicates the thrombotic terminal vein of the left side; (*h*) fresh hemorrhage of the cerebral white matter produced by confluence of capillary hemorrhages; (*s*) discoloration of the periventricular area indicating involvement of the white matter; (*t*) thalamus.

filtrating the subependymal area of the caudate nucleus; hemorrhages, which originate at the confluence of the anterior and posterior terminal veins, destroying the body of the caudate nucleus; hemorrhages, which arise from the posterior root of the terminal vein, involving the narrow tail of the caudate nucleus.

Hemorrhage from the terminal vein is commonly bilateral but in general it is more marked on the right side. The extravasated masses often destroy large areas of the caudate nucleus. Hemorrhages originating from the stem of the terminal veins are usually associated with bleeding from their intracerebral roots, draining the cerebral white matter and the basal ganglia. In such cases

groups of punctiform and strip-like hemorrhages of the frontal and fronto-parietal white matter radiate from the angles of the lateral ventricles in the direction of the periphery of the hemispheres.

Lesions of the *ventricular lateral vein* frequently occur in the newborn child. The stem of this vessel is generally not involved; the changes affect the cerebral area drained by this vein, namely, the occipital white matter; strip and punctiform hemorrhagic foci appear which often form massive hematomas. (Fig. 4). Another frequent combination of birth traumatic circulatory disturbances is hemorrhage from the choroidal vein, completely filling the lateral ventricles.

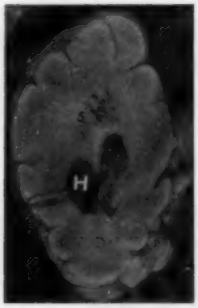


Fig. 4. Massive hemorrhage and multiple softening foci in the subependymal white matter of the occipital lobe in the region supplied by the lateral ventricular vein. Full term baby, 3-day-old. (*s*) fresh softening foci; (*h*) massive hemorrhage; (*p*) choroidal plexus.

Apart from these frequent and extensive intracerebral hemorrhages, the brain of a newborn infant almost regularly shows microscopic extravasations located in the corpus callosum, the basal ganglia, mid-brain, medulla oblongata, cerebellum, retina, and in the inner and middle ear; that is, in all those parts of the brain in which the venous drainage is connected with the vein of Galen and, therefore, is susceptible to the lower-pressure-effect.

The circulatory disturbances produced by the suction effect of the lower-pressure-principle are not always characterized by extravasations of blood. In many instances there is only extreme dilation of the vessels which are engorged with blood, demonstrating a circulatory paralysis. This stasis of the blood develops in the

same areas — namely, in the frontal, fronto-parietal and occipital white matter — which are known to be affected by the hemorrhages, that is in the areas drained by the lateral ventricular and terminal veins. Consideration of the nonhemorrhagic circulatory disturbances is of utmost importance in the understanding of many conditions occurring in the pathology of the newborn.

BIRTH TRAUMATIC SOFTENING PROCESSES IN THE CENTRAL NERVOUS SYSTEM OF THE NEWBORN

Two types of birth traumatic cerebral softening processes can be distinguished in the newborn: (1) *Circumscribed foci of necrosis and dissolution* of the nervous tissue; and (2) *diffuse rarefaction*. Foci of necrosis may be detected as early as 24 to 48 hours after birth. Those small grayish-yellow foci are always located in the areas of the brain which we have recognized as typical sites of circulatory disturbances resulting from birth trauma (Fig. 3s, 4s). In children who survive, the foci become progressively denser and whiter in appearance. Characteristic of the foci in older children is that they develop a shining chalk-white color and be-

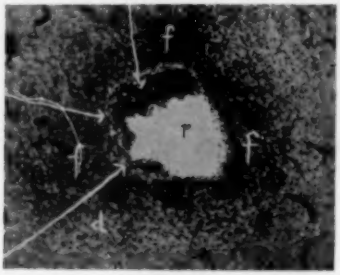


Fig. 5. Microscopic aspect of a birth traumatic softening focus of the cerebral white matter. Beginning granulation stage in a 10-day-old child. (Scarlet-red-hematoxylin-staining). P: indicates a small hollow developed in consequence of the dissolution of the necrotic center of a birth traumatic necrosis. Arrows: indicate the demarcation zone of the damaged cerebral tissue, in which the glial proliferation proceeds. *f.f.f.*: indicate the zone of the softening focus, which is characterized by the accumulation of fat-laden scavenger cells. *d.d.d.*: indicate the environment of the softening focus characterized by diffuse rarefaction of the nervous substance.

come rather hard. The first few days following birth, necrosis and disintegration predominate microscopically; 6 days to 10 days after birth evidence of glial proliferation appears, surrounding and

localizing the destruction. (Fig. 5). If death occurs some weeks or months after birth, cicatrization prevails, characterized by the progressive accumulation of glial fibers.

Microscopic foci of cerebral softening occur very frequently in the newborn. They have been demonstrated at postmortem examination in almost every child surviving the first day, and in those who have been examined during the first four weeks. Circumscribed foci are regularly associated with diffuse softening processes.

The fatty substances which characterize the cerebral softening in the newborn can be demonstrated histologically using the scarlet red stain. In addition to this fatty debris, however, there are other signs of tissue damage present — such as hemorrhages, fragments of nerve fibers, disintegration of the glial nuclei, and reactive proliferation of the glial cells. These changes are identical with processes observed in adults following circulatory disturbances.

FAT DEPOSITS IN THE GLIAL CELLS OF THE NEWBORN

Virchow (1868) discovered that the presence of fat granules in the glial cells of the newborn was a frequent occurrence. He considered these findings to be characteristic of an "encephalitis" due to an intra-uterine infection which had been transmitted to the fetus from the mother. Jastrowitz (1870, 1872) however disagreed with this view and maintained that the fat deposits in glial cells were of physiological significance. He assumed that they were in some way connected with the development of the myelin sheaths. It was his belief that those very rare cases revealing an absence of fatty glial cells represented disease of the fetal central nervous system. This opinion dominated for almost half a century. Eventually this doctrine became modified in the following way: certain fatty glial cells were regarded as of physiological significance, others were considered to be characteristic of pathological processes. Thus, Merzbacher (1907) discriminated two types of fatty glial cells in the brain of the newborn: (1) Round, physiological elements, resembling genuine phagocytes which transport fatty substances to the developing myelin sheaths; (2) Fatty glial cells with extensions which are anchored within the nervous substance, signifying a morbid condition. Wohlwill (1921) also tried to differentiate physiological fat-containing glial elements from pathological scavenger cells. He considered, however, the cells having

extensions to be physiological and regarded the elements which Merzbacher described as physiological to be of pathological significance. All these attempts to differentiate the fat-containing glial cells have no true practical value, because, generally both types of cells occur together. Therefore, Merzbacher would, by a strict application of his criteria, designate the same number of cases as being pathological, as would Wohlwill, in spite of the fact that Merzbacher considered Wohlwill's physiological elements to be of pathological significance. On the other hand, both, Merzbacher and Wohlwill, would have to accept the same incidence of pathological cases as Virchow claimed, who considered all fatty glial cells to signify disease.

The doubtfulness of the significance of the glial fat deposits in the newborn was so deeply rooted that any consideration of the pathological changes of the central nervous system in newborn infants could not be made without previous clarification of this problem. Investigations carried out by the present writer revealed that fatty elements normally do not occur in the cerebral substance of the newborn. Thus we confirmed Virchow's teaching. Also, our experience showed that *the problems which arose as a result of Virchow's discovery are identical with the questions posed by the birth injury of the newborn.*

It is of importance that in addition to fat deposits in the white matter, in the same areas in which the glial fatty changes prevail, other signs of morbid changes are present almost without exception, e.g., hemorrhages, fragmentation of nerve fibers, disintegration as well as hypertrophy and proliferation of the glial cells. The significance of these changes cannot be doubted. If nothing were known of the presence of the fatty deposits in the brain, then these other changes alone would be more than sufficient to characterize morbid conditions affecting the brain of newborn infants. It should be emphasized that all these changes are invariably associated with glial fat deposits, in the diseased nervous system of adults. The fatty glial cells observed in the brain and spinal cord of adults are identical in every way with the controversial phenomena occurring in the central nervous system of the newborn. Furthermore, it is important to know that the brain and spinal cord of animals — cattle, rats, rabbits, dogs, and cats were investigated by us — develop without the appearance of any fatty elements whatsoever. However, identical changes to those seen in the

brain substance of the newborn child, including fat-containing cells, have been experimentally produced in the brains of newborn animals by traumatic lesions.

LATE SEQUELAE OF BIRTH TRAUMATIC LESIONS OF THE CENTRAL NERVOUS SYSTEM

Many cerebral defects observed in older children and adults may be explained by birth injury. Such defects occur very often in persons suffering from feeble-mindedness, cerebral palsy, and epilepsy. The following are the main types of brain damage which we have observed in these cases at postmortem examination:

1. Cavitation of the brain (so-called central and peripheral porencephaly).
2. Contraction of the cerebral hemispheres caused by cicatricial hardening (sclerosis of the white matter).
3. Cicatricial shrinking of the cerebral cortex (sclerotic microgyria).
4. Cicatricial defects of the basal ganglia, particularly of the caudate nuclei.
5. Net-like cicatricial strips characterized by excessively myelinated irregular nerve fibers (status marmoratus of the striatum and the thalamus; plaques of myelinated nerve fibers in the central cortex).
6. Hemorrhagic internal pachymeningitis.
7. Internal hydrocephalus.

All these lesions may occur together or in a variety of combinations. Cavitation and cicatricial contraction may also occur in the cerebellum (cerebellar porencephaly and sclerotic cerebellar atrophy). Cavities in the medulla oblongata (syringo bulbia) or in the spinal cord (syringo myelia) represent rarer defects resulting from birth trauma.

In these cases cavitation deep within the cerebral hemispheres is caused by a dissolution of the white matter in the area drained by the terminal and lateral ventricular veins. (Fig. 6). In those instances in which changes are not of long standing, hemosiderin pigmented masses may be found at autopsy representing remnants of the initial circulatory disturbances.

Cicatricial contractions (sclerosis) of the cerebral white substance also develop in the area drained by the terminal and ventricular lateral veins. This type of lesion is the result of processes

characterized by the destruction of nerve fibers and by diffuse rarefaction of the glial structures. Sometimes the loss of nervous parenchyma is so excessive as to almost reach complete devasta-

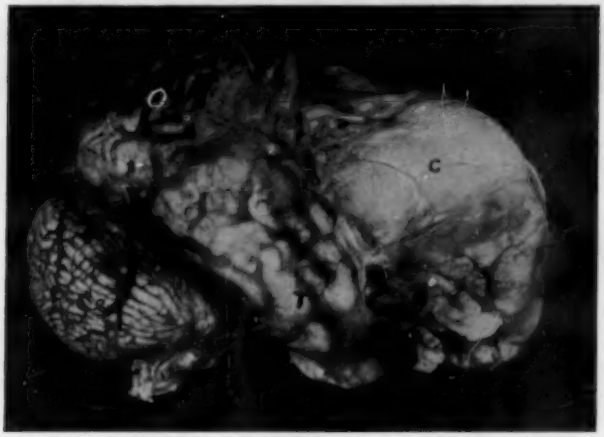


Fig. 6. Extensive porencephalic transformation of the right cerebral hemisphere in an idiotic child. All those areas of the white matter which are supplied by the terminal vein and the lateral ventricular vein have disappeared. Extensive parts of the cerebral cortex are also absent, having been destroyed by birth injury, save for a very thin molecular sheet covered by the pia ("vesicular porencephaly"). The cortex of the occipital lobe is shrunken ("sclerotic microgyria"). There are also cavities in the left hemisphere, but a shrinking of the white matter caused by diffuse scarification dominates. One-and-a-half-year-old girl, full term child, first born; delivery lasted two days and was accomplished by forceps. The child exhibited cramps immediately after birth. At autopsy, complete ossification of the sagittal suture was disclosed; the pia was covered by hemosiderin and the head of the caudate nucleus was absent on both sides. (*c*) vesicular transformation of the frontal end of the parietal lobe; (*o*) sclerotic microgyria of the occipital lobe; (*ce*) cerebellum; (*t*) temporal lobe.

tion. No doubt, severe birth traumatic rarefaction processes involving the fronto-parietal and occipital white matter can produce similar functional disorders, for example, idiocy and paralysis, as the dissolution of the nervous tissue. The functional significance of the less severe grades of birth traumatic rarefaction processes which involve the cerebral white matter have not been sufficiently investigated despite their frequent occurrence. We assume that less severe grades of feeblemindedness as well as many cases of so-called psychopathy and neuropathy in children and adults may be caused by this type of injury.

The entangled masses of myelinated irregular nerve fibers which

represent the most striking morphological aspect of the ***status marmoratus*** in the striatum and thalamus may often be recognized with the naked eye; while those cortical coils, the "plaques fibromyéliniques", are only demonstrable microscopically. Marked status marmoratus of the striatum is regularly associated with characteristic athetoid motility disorders. In some cases of status marmoratus of the thalamus we observed peculiar disturbances of affect. A status marmoratus of the basal ganglia develops following birth traumatic rarefaction processes involving areas drained by the terminal vein.

Internal hemorrhagic pachymeningitis frequently accompanies contraction of the cerebral hemispheres of birth traumatic origin. Extensive pial hemorrhages at birth characterize the beginning of this condition. During the later development of the process, however, new pial hemorrhages occur. Another typical feature is the appearance of thick membranes covering and enveloping the pial hematomas.

Internal hydrocephalus caused by birth trauma is often a rapidly progressing disease. It may develop as a result of the occlusion of the aqueduct by blood clots or the obstruction of the circulation of the cerebrospinal fluid, following hemorrhages surrounding the medulla oblongata and the cerebellum.

In our investigations we encountered a large number of cases in which various cerebral defects were caused by genic germinal disease or resulted from infectious, toxic, or traumatic lesions suffered during intra-uterine life. These observations prove that true innate diseases of the central nervous system, of hereditary and nonhereditary origin, do occur. Also, there can be no doubt of the occurrence of extensive brain lesions caused by infectious and toxic agents or trauma endured in the early weeks or months following birth. Nevertheless, it is pertinent that *clinical symptoms indicating severe involvement of the central nervous system which can be observed from the first day of life, or generally speaking, which appear in the first year of life — perhaps following a period of apparent well being — are caused in the great majority of instances by birth injuries.*

REMARKS ON TYPICAL EXTRACEREBRAL LESIONS IN NEWBORN CHILDREN

In addition to the lesions of the central nervous system resulting

from physical trauma above described, hemorrhages and necroses in various organs of the newborn occur which we consider to be indirect results of the cranial birth injury. These changes, in general, affect children who die a few days following birth or who die during birth after remaining for an abnormally long time in the birth channel subsequent to the rupture of the amniotic sac. The most frequent pathologic-anatomical findings in this group are represented by hemorrhages and necroses of the adrenal glands, which appear both in premature and in full term children. Also, renal involvements occur strikingly often. They consist of necroses of the tubular epithelium and hemorrhages, resembling in every detail changes found in the so-called crush syndrome. Particularly interesting are cases in which selective hemorrhagic infiltration of the medulla of one or of both kidneys is present. Here we must mention hemorrhages and necroses of the liver in newborn children, which are similar to lesions observed in patients who died of puerperal eclampsia or following shock. Furthermore, hemorrhages of the spleen and pancreas as well as the thymus, the thyroid gland, and the parathyroids were repeatedly described. In particular, mention must be made of pulmonary hemorrhages which, rather frequently, may arise giving the impression of a hemorrhagic infarction. Hemorrhagic erosions of the gastric mucosa and — though much more infrequent — duodenal ulcers or melena caused by innumerable mucosal lesions of the intestines, form a special group. Necroses of the heart muscle appearing subsequent to the birth were also observed (Grünwald).

We may assume that all circulatory disturbances accompanied by hemorrhages and necroses described here, result from birth damage to cerebral and spinal centers which regulate the blood supply of the listed organs. It is very remarkable that many of the visceral lesions described here also occur in adults, who have suffered severe traumatic cerebral lesions.

Another interesting group of lesions in the newborn concerns the damage to regulatory centers of the peripheral circulation. Symmetrical circulatory disturbances of the hands and feet appear as in Raynauds' disease in adults. Localized or generalized edema of the body is also a condition which draws attention to vasomotor disturbances in children. Two other enigmatic disorders in the newborn, sclerema and subcutaneous fat necroses which are sometimes linked to one another, have been thought to result from a

local mechanical stress; but obviously the part played by the general stress at birth should also be considered.

REMARKS ON THE RELATIONSHIP BETWEEN BIRTH TRAUMA AND THE PATHOLOGY OF THE NEWBORN

Clinical investigation stimulated by our anatomical findings revealed that it is necessary to consider newly born children from the viewpoints which dominate the physiology and pathology of adults. In accepting this attitude, we recognize physiological standards of the healthy newborn which in many ways are comparable with those of healthy adults. Consequently, deviations from these standards in newborn children are to be considered as manifestations of morbid conditions as they would be in adults. Such deviations include many peculiar differences in the behavior of various newborn children which until now were generally explained by the immaturity of the newborn.

The assumption that the immaturity of the central nervous system of the newborn was responsible for many phenomena which in adults would be considered as symptoms of severe brain injuries was so deeply rooted that even convulsions, which differ in no way from those in adults, were interpreted as of physiological origin. Recent investigations, however, have shown that there is no evidence for making such a paradoxical hypothesis. Convulsions in both children and adults have the same significance — namely, cerebral disease.

Certain well-known neurological signs, such as pathological reflexes, spontaneous movements of the hands and fingers, convulsions, paralysis, flaccidity or rigidity of the skeletal musculature, unconsciousness, stupor, or dizziness can be observed in adults who have suffered cerebral damage; they may also be demonstrated in newborn infants who have endured similar injuries due to lesion at birth. In addition to these well-known effects, spontaneous nystagmus and disorders involving the rotatoric and caloric reactions of the vestibular apparatus often occur in both adults and newborn children who suffered cerebral injury. (Voss; Berberich). Furthermore, typical changes of the morphologic compositions of the blood are of diagnostic importance; in addition, bilirubinemia, transitoric albuminuria, and glycosuria, and also disturbances in temperature regulation may be present in the traumatized newborn infant.

It is unnecessary to stress that certain respiratory difficulties and "debility" observed in newborn children are, in many cases, symptoms of brain injuries.

As in adults, lumbar puncture in newborn children is not only of diagnostic importance in traumatic cerebral hemorrhage, but it is also of therapeutic benefit. (Sharpe).

REMARKS ON THE PREVENTION OF BIRTH INJURIES OF THE CENTRAL NERVOUS SYSTEM

Birth trauma as a factor in the mortality rate of infants and newborn children has been shown by extensive statistical investigations to be a social problem. (Schwartz, Baird). Prematurity, which is responsible for the large majority of deaths occurring at birth and within the first months of life, represents a phenomenon of poverty which must disappear with the rise of the material and intellectual social level.

We concluded that it was necessary for services to be created which could safeguard expectant mothers and allow them to carry their children to full term. An organization which cares for all pregnant women and ensures that every birth is supervised by adequately trained personnel would undoubtedly lead to a reduction of the mortality rate due to birth lesion. Also, we are certain that social preventive measures could dramatically reduce many of the later consequences of birth injury — particularly cerebral palsy, feeble-mindedness, and epilepsy.

In addition to social preventive measures, a reform of obstetrical management is imperative. The birth which is easy for the mother is not of necessity without danger for the child. In order to estimate this danger to the child it is necessary to continually register the intra-uterine pressures which develop before and during birth. In this way the limits of the pressure differences which the child can tolerate without danger will be defined. Such observations may result in more accurate indications for operative interference in obstetrics.

In view of the importance of the lower-pressure-principle as the main cause of birth traumatic lesions of the newborn, the following conclusion may be drawn: *Whenever abnormalities of the birth canal, the placenta, malpresentation of the fetus or disproportion between fetus and pelvis indicate that normal birth might be injurious to the child, Cesarean section performed before the begin-*

ning of the labor and before the rupture of the amnion sac is the best recommendation.

COMMENT

The danger to which the child is exposed during the birth process has been known for centuries and has been the subject of scientific investigations for almost 200 years. The fact that stillbirth is commonly caused by cerebral damage, and that a birth injury may be the cause for lasting deformation of the body and impairment of the mental function was also well known. As early as a century ago Little stated that "congenital" cerebral palsy was caused by birth trauma in the majority of cases; and as early as 125 years ago Cruveilhier recognized that the "apoplexy" of the newborn could be prevented, in the great majority of cases, by proper obstetrical measures.

Birth lesions and their results have been described in thousands of publications during the last hundred years. *With the increasing number of births throughout the world, there is an important increase in the number of victims every year.* Many of them die at birth or during the first weeks of life; however, innumerable survivors remain as paralytics, morons, idiots, and epileptics.

Why is it that birth lesion of the newborn, in contrast to other big and smaller stranglers of humanity — such as tuberculosis, cancer, infantile paralysis, and multiple sclerosis — is still not recognized as one of the important causes of untimely death, invalidism, and chronic suffering? Why is it then that no systematic measures have been proposed to fight the birth injury of the newborn?

These questions have a simple answer, viz., the main cause is the fact that the birth lesion of the newborn represents a social problem. Its victims — premature children, illegitimate children, children of mothers who have a contracted pelvis, first-born children of older women, children born without help or competent assistance — belong to the economically weak groups of the population in the great majority of cases.

All contemporary communities are developed on the basis of keen competition in which a relatively small group of successful people live with a large mass of people persisting in insecurity. Thus, it is necessary that a community attain a certain level before it is able (1) to accept with good conscience the connection between

birth injury and socio-economic conditions; (2) to acknowledge that the loss of human life and the limitations of mental and physical efficiency resulting from birth lesions are a heavy burden to the community; and (3) to be ready to apply remedial measures.

When every woman will be enabled as a young person to deliver her first child, when every pregnant woman will be permitted to carry her child to the end of the normal term, and when every birth will be supervised by adequately trained personnel, then the danger of the birth lesion will be reduced to a minimum.

There are some other points to be considered. (1) Most autopsy reports on newborn infants only consider extravasations into the pia and the dural folds, in spite of the fact that the present writer and some of his supporters emphatically stated the presence of important foci of hemorrhage and softening in the brain substance proper. Thus, we have to admit that we have not been successful, as yet, in establishing it as an omission or even as a failure of observation when a physician does not register the macro- and microscopic cerebral changes in a case of birth traumatic lesion. (2) The discussion on the significance of the glial fat deposits in the central nervous system of newborn children lasted a very long time. Eventually, the main participants in the new discussion — among them authors who at first dissented — agreed on the pathological significance of the findings and on their birth traumatic origin in most cases. In spite of these facts, we have not succeeded in bringing to general attention the knowledge that the changes discovered by Virchow represent the most frequent, and in many cases a very serious disease of the human central nervous system. (3) Our clinical investigations performed with Voss and Berberich on the vestibular apparatus resulted in many new observations, which together with the more conventional neurological symptoms, demonstrated the frequency, unexpected in earlier times, of the birth traumatic cerebral lesion in newborn infants. Although repeatedly verified, these methods and observations are still not generally known, and, therefore, newborn children who suffered brain damage still are not considered as subjects for competent neurological examinations. (4) In serial investigations Sharpe showed that lumbar puncture in newborn children is both informative and safe. However, this test is still not as widely used as it merits. (5) We have shown that changes of the morphological blood composition (the "birth traumatic blood crisis of

the newborn") as well as the bilirubin and uric acid levels in blood are sensitive indicators of the destruction of blood and tissue, and, therefore, are suitable gauges of the intensity and the extent of the damage resulting from birth trauma. As a result of brain damage, disorders of temperature regulation often occur in newborn infants. Albuminuria and glycosuria in newborn children, may also indicate central disturbances of metabolism. In addition, pathological and anatomical observations have revealed that renal involvement often occurs in newborn infants. We have to recall here the high frequency of adrenal lesions in newborn children. All these findings, often observed by numerous investigators, have not been able as yet to influence the clinical pathology of the newborn. (6) One of the most important causes of the delay in the general recognition of the importance of cerebral birth injury in the growth, in the composition, and in the quality of the population, is the fact that the interpretation of the birth mechanism, made possible by the definition of the lower-pressure-principle, is not generally known, although it is acknowledged by competent experts and endorsed in experiments.

A study of the modern history of the birth mechanism revealed that the writer was not the first to assume that the birth process obeys the laws of hydrodynamics. (Schatz in 1868, Kuneke in 1869, Lahs in 1869, Werth in 1888, Sellheim in 1913). Sellheim's remarks in the discussion of a report by the writer, in 1926, even today seems suitable to describe the significance of the lower-pressure-principle as well as the degree of its recognition by obstetricians: "I struggled for a long time for the recognition of the importance of the hydraulic pressure. Only a few obstetricians were helpful in this endeavor. Then verification came from another side after observing new facts, i.e., the effect of the hydraulic pressure on the cerebral blood vessels. Pathologists and pediatricians have spoken of hemorrhages caused by a suction effect during birth."*

"These observations verify not only the role of the hydraulic pressure but also the correctness of my didactical endeavor to characterize the pressure differences between the uterus contents and the atmosphere during birth as a suction effect of the atmosphere."**

*The heading of the present writer's first paper (1921) on birth traumatic brain injury was: "Die Ansaugungsblutungen im Gehirn Neugeborener" (*Cerebral Hemorrhages in Newborn Children Caused by Suction Effect.*)

**This didactically much more effective interpretation was published by Sellheim in 1923.

"The concept of a suction from below replacing the idea of a pressure from above may explain all the peculiar phenomena of birth from the stretching of the child's body in utero to the cerebral hemorrhages."

A most important experimental contribution to the problem of the lower-pressure-effect was that of Guttner (1932). He tried to imitate the birth machine and used:

1. A relatively large glass jar with a narrow neck representing the uterus.
2. A rubber sac which, replacing the amnion sac, was pulled into the "uterus."
3. A guinea pig which was placed in the system in a manner imitating the normal birth posture.

The construction of the apparatus permitted the elevation of the "intra-uterine" pressure by pumping in air and also the measurement of the changes in blood pressure and the heart function of the "fetus."

The results of these experiments were extremely interesting. While the entire body of the animal remained in the closed rubber sac under increased pressure, no changes occurred either in the blood pressure or in the heart function. However, when the head of the guinea pig became fixed in the "pelvis"—in imitation of the natural process—and the rubber amnion was torn, increase of the "intra-uterine" pressure caused a rise of the arterial tension, in addition to characteristic changes in the heart function, similar to those observed in natural birth. Thus, Guttner succeeded in reproducing some factors of normal delivery by creating pressure differences between "uterus" contents and the atmosphere in a system in which the head of a living animal closed the "os uteri," and then was released from the high pressure which affected all other "intra-uterine" parts of the body.

In our own experimental investigations, repeatedly verified by other authors, a suction cup was placed on the heads of newborn animals. With this simple method we were able to produce all cranial and cerebral changes occurring in normal and pathological births.

We hope that when the lower-pressure-principle becomes generally known and acknowledged by obstetricians as the most important driving force of the birth mechanism, it will then become generally recognized that lesions of various degree almost always

occur in every natural birth. At the same time, we hope, that methods will be devised which will neutralize the detrimental effect of the pressure differences.

BIBLIOGRAPHY

Baird, D.: Difficult Labor, It's Incidence, Prevention and Management. Obstetrics, p. 525, Philadelphia, 1955.

Berberich, J. and Wiechers, A.: Zur Klinik des Geburtstraumas. Ztschr. f. Kinderh., 38:59, 1924.

Ehrenfest, H.: Birth Injuries of the Child. New York, 1931.

Guttner, M. D.: Über das Verhalten der Blutzirkulation des Fetus in der Geburt. Arch. f. Gynäk., 149:70, 1932.

Holland, E.: Birth Injury in Relation to Labor. Am. J. Obst. & Gynec., 33:1, 1937.

Schwartz, Ph.: Die Ansaugungsblutungen im Gehirn Neugeborener. Ztschr. f. Kinderh., 29:102, 1921.

Schwartz Ph.: Die traumatische Gehirnerweichung des Neugeborenen. Ztschr. f. Kinderh., 31:51, 1922.

Schwartz, Ph.: Erkrankungen des Zentralnervensystems nach traumatischer Geburtsschädigung. Ztschr. f. Neur. Psychiat., 90: 263, 1924.

Schwartz, Ph.: Die geburtstraumatische Schädigung des Kopfes Neugeborener und ihre Bedeutung fur die Pathologie. Monatschr. f. Kinderh., 34:511, 1926.

Schwartz, Ph.: Die traumatischen Schädigungen des Zentralnervensystems durch die Geburt. Anat. Untersuchungen. Ergebn.Inn. Med. u. Kinderh., 31:165, 1927.

Schwartz, Ph.: Geburtshilfe und Hirnschädigungen durch die Geburt Zentralbl. f. Gynäk., 52:2116, 1928.

Schwartz, Ph.: Birth Injuries of the Newborn. Investigations Into Their Morphology, Pathogenesis, Clinical Pathology and Prevention. Monograph, to be published.

Sellheim, H.: Die Geburt des Menschen. Leipzig, 1913.

Sellheim, H.: Remarks on Birth Trauma. Zbl. Gyn., 50:3359, 1926; Monatschr. f. Kinderh., 34:617, 1926.

Sharpe, W.: Intracranial Hemorrhage in the Newborn. Am. J. Obst. & Gynec., 9:452, 1925.

Sharpe, W.: Neurosurgery in the Newborn and Children. New York State J. Med., 27: 296, 1927.

Voss, O.: Klinische und patholog.-anatomische Folgeerscheinungen geburtstraumatischer Schädigungen des Felsenbeines. Monatschr. f. Kinderh., 39:568, 1926.

Voss, O.: Geburtstrauma und Gehororgan. Acta oto-laryng., 11:73, 1927.

PULMONARY INFILTRATION WITH EOSINOPHILIA DURING ISONIAXID TREATMENT. P. Perreau, M. Fresnau, B. Boumard and Y. Le Brizaut. (Presse méd., 63:1454-1456, Oct. 29, 1955).

The authors report two cases of labile pulmonary infiltration with eosinophilia in patients with tuberculous meningitis that was being treated with isoniazid. One patient was a 30-year-old woman in the eighth month of her second pregnancy, and the other a 5-year-old boy. Treatment had continued uneventfully over a period of several months before the appearance of the reactions, the drug being administered orally in doses of 5 mg. per kilogram. The infiltrate made its appearance several weeks after elevated doses had been started, that is, 20 mg. per kilogram. It was accompanied in both cases by digestive and blood disturbances of the kind seen in other patients treated with high doses of isoniazid, which fact suggests that drug intoxication was the cause of the pulmonary infiltration with eosinophilia. J.A.M.A.

CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish from time to time papers from the classes of Medical Colleges and Universities. Interested medical schools are cordially invited to submit student papers for consideration.*

BACTERIOLOGICAL COUNTS ON INFANT FORMULAS MIXED IN THE HOME*

DONALD B. LATHROP
New York.

Much experimental work has been done on culturing infant formulas to make certain that the milk given to infants is bacteriologically safe. Most of this work has been done in hospital formula rooms, the conclusions being that terminal sterilization is the best method of preparing large amounts of daily formula for hospital nurseries.[1, 2, 3]

Little, if any, experimental work has been done on formula preparation in the home. A thorough investigation has revealed no published work in the medical literature, and only a few unpublished reports from various sources.[4, 5] Extensive work has not been thought necessary as there are any number of pamphlets on "infant care" which are liberally supplied to new mothers giving explicit instruction in the terminal method of sterilization as applied to conditions in the home.[6, 7, 8] This procedure appeared to have been satisfactory as applies to mothers who are planning to bottle-feed their babies. This group make up a full one or two day's supply of formula at one time, sterilize the formula terminally, and can then refrigerate it until its use. This is convenient to the mothers, and has turned out to be satisfactory for all concerned.

However, there is another group of mothers who make up infant formula whose needs are different from those who entirely bottle-feed their babies. These are the women who breast-feed their infants, and who need to give the baby a supplemental bottle from time to time, because their own milk supply is not quite sufficient for the infant's needs. Such a situation is common in many women

*Submitted as partial fulfillment of the requirements of the course from the classes of Dr. Walsh McDermott at the Cornell University Medical College, New York.

throughout the breast-feeding period, and is especially frequent in the days when the mother's milk flow is drying up, and the mother wishes to wean the baby to a cup. At such times, when the mother needs to mix only one bottle per day or every other day, it is impractical to use a terminal sterilization method on every bottle. It is easier to mix just one bottle at a time and feed this to the infant. For this single bottle mixing, it may be convenient to the mother to use one of the prepared infant formula mixes, of a composition closely approximating human milk. This milk is available commercially in both powdered and liquid forms; the liquid formula is quicker to prepare, while the powdered preparation can be stored longer, a minimum of contamination occurring even after the can is opened.

A number of problems are presented in this feeding situation, problems which are important to the mother, but which do not lend themselves easily to scientific investigation. Should the water and formula bottles used be sterilized, or is a good washing sufficient? Once the formula has been prepared, how long can it be kept under refrigeration before it is unsafe for the baby to drink and, therefore, should be discarded? If an infant drinks only a portion of his bottle, can the remainder be kept and fed to him again at a later time, or, must it be thrown out, because of regurgitation contamination by the baby when he drank the first portion?

In an effort to investigate some of these problems, we asked a number of women to mix formulas for us, and then we cultured the milk at intervals, before and after a baby had drunk from the bottle, in an attempt to study the bacterial contamination of the milk at the different times.

II. METHODS

Regular eight-ounce infant formula bottles were used. They were filled with water, the nipples inverted and placed on top, the bottles capped, and the combination boiled in a bottle sterilizer for fifteen minutes. The sterilized bottles were then refrigerated until needed. Powdered Similac was used to make the formula in all instances. It was found that this brand of powdered milk had an extremely low bacterial count per gram of dry powder.[9, 10] This gave us a sensitive base line from which contamination of the mixed formula by mother or infant was readily revealed. The powdered milk, in

new, unopened cans, was supplied to the women who were mixing the formula. Buried in the milk powder of each can, was a "measuring spoon"; four measurefuls were added to eight ounces of water to prepare the formula. The procedure followed by the women was as follows:

1. the Similac can was opened, and the measuring spoon dug out of the milk in whatever way seemed suitable to the woman subject;
2. the cap and the nipple were removed from the bottle and placed to one side, in whatever way seemed suitable to the subject;
3. four measures of powdered milk were transferred to the sterile baby bottle containing eight ounces of sterile water;
4. the cap and nipple were replaced; the mixture was shaken until the powdered milk went into solution;
5. the prepared formula was refrigerated immediately.

The water could be heated or not, to aid easy solution of the powder, depending on the women's preference.

A questionnaire was circulated after the experiment was terminated in an attempt to determine exactly how each woman mixed the formula. Women only were selected who were not laboratory or hospital personnel trained in aseptic technique. They all had had children of their own at some previous time, and all lived under reasonably clean conditions in the home.

After preparation, the formulas were used for experiment as described below. During each experimental step, controls were used where necessary to measure outside contamination.

Plating of the formula was done according to Standard Methods[11] on Bacto Plate Count Agar (Tryptone Glucose Yeast Agar).*

All plates were incubated at 37°C. for 72 hours, and then checked by the plate-count method. No attempt was made to

*Composition of the Plate Count Agar

Formula	*Per. Liter*	
Bacto Yeast Extract	2.5 gm.	
Bacto Tryptone	5.0	
Bacto Dextrose (Glucose)	1.0	
Bacto Agar	15.0	
TOTAL	23.5 gm.	dehydrated agar added to 1000 cc. water
Final pH 7.0		

classify the bacteria plated, although selected colonies were gram-stained and inspected microscopically. Five plates were incubated at 15°C. for 3 days to 5 days in an attempt to isolate any thermophobic bacteria, but no cultures grew in any of these plates.

III. RESULTS

Part 1: The formula of powdered Similac and sterilized water was mixed, refrigerated for 48 hours at approximately 10°C. (50°F.), and plated on the Tryptone Glucose Yeast Agar. Incubation was for approximately 72 hours, and the plates were counted.

Table 1.

Formula mixed by:	*Count per cc.*
H	0
L	2
L	3
L	22
M	13
Me	480
Me	1,300
Me	2,000
T	14
T	27

In general, the formulas mixed were relatively free from bacteria. However, it can be seen that one woman, Me, consistently contaminated her formula. The reason for this may be seen from the results of the questionnaire, indicating the methods used by each of the women in mixing the formula. (see Table 2).

The steps in mixing the powdered milk and water actually allowed little chance of contamination. Each can of Similac was new and unopened. The woman mixer had to open the can with a turnkey, which did not allow any contact between the turnkey or the mixer's fingers and the powdered milk. The greatest possibility of contamination came in the next step; the mixer had to dig down under the surface into the powdered milk in some way to secure a measuring spoon buried therein. Four of the women, whose formulas were relatively uncontaminated, dug the measure out with a clean but non-sterile utensil, avoiding any contamination of the milk with their fingers. However, the fifth mixer, Me, admitted that she had dug into the powdered milk with her fingers

Table 2.

Questionnaire Submitted to Formula Mixers

Material supplied:
1) Sterile bottle with 8 oz. sterile water therein.
2) Unopened can, powdered Similac.

Directions:
1) Open Similac can, take out measuring spoon.
2) Place 4 measures powdered Similac into sterile bottle and mix.
3) Refrigerate mixed formula immediately.

Procedure:	H	L	M	Me	T
1) Did you wash your hands before mixing the formula?	yes	sometimes	don't remember	yes	yes
2) After opening Similac can, how did you obtain the measuring spoon, which was buried in the powdered milk?	dug it out out with a knife	dug it out with a knife	dug it out with a spoon	scooped it out with my fingers	dug it out with a spoon
3) In measuring out the powder, how did you obtain an approximate measureful?	scraped off excess	scraped off excess	scraped off excess	tapped off excess	scraped off excess
4) If the excess was scraped off, with what did you scrape it off?	knife	knife	knife	tapped it off	knife
5) How did you transfer the powdered milk to the bottle?	scraped it in with a knife	scraped it in with a knife	dropped it in slowly	dropped it in slowly	through a funnel
6) If any powdered milk spilled, did you scoop it up and place it in the bottle?	no	no	none spilled	no	no
7) Where and in what position did you place the nipple and bottle cap when filling the bottle?	on clean towel nipple up	on clean surface with nipple up	on clean towel nipple up	on table nipple up	on table nipple up
8) How soon after mixing was the bottle refrigerated?	immediately	immediately	immediately	immediately	immediately
9) Did you heat the water while mixing?	yes	yes	yes	no	no

in order to secure the measure; this would provide an easy source of contamination, even if the mixer had previously washed her hands. To complete, one must transfer four measures of powder to the bottle, replace the bottle cap, and shake until mixed. The only other easy source of contamination here, aside from mishandling or dropping the nipple and cap, was if any attempt had been made to scoop up any spilled powder and add it to the bottle —none of the mixers did this. Whether or not the water was heated during the process does not seem to make any significant difference in the bacterial count; all mixers refrigerated the mixed formula immediately.

Therefore, we may conclude, from the results stated, that the contamination of the formula by Me was due to her finger contact with the milk.

Another aspect of the contamination of the formula by the mixer was to place chromogenic bacteria on the mixer's hands, prepare the formula allowing no direct contact between hands and milk. Then we checked how much contamination of the formula was due to the mixer by counting the chromogenic colonies plated. This experiment was tried using *B. prodigiosus* as the chromogenic bacterium; it was tested previously, to verify that the strain grew well on the Tryptone Glucose Yeast Agar, producing light pink colonies. The results are shown in Table 3.

Table 3.

Mixer	*Count*	
	1cc.	*0.1cc.*
L	0	0

No contamination was observed at all on this single attempt, and further investigation was abandoned.

Part 2: Another aspect of the problem facing mothers who mix formula for their infants is that of contamination of the milk by regurgitation by the infant while drinking it. If the infant drinks only a portion of the bottle, how long can the rest be kept and refed to the infant, before bacterial growth makes the formula unsafe for drinking?

To investigate this problem, we did two sets of cultures. In the first, the infants drank one ounce and four ounces of milk out of different bottles. These bottles were left at room temperature for one hour after drinking, and were then plated. It can be seen

that the amount of contamination varied according to the amount the infant drank from the bottle, or rather, according to the amount the infant regurgitated back into the bottle. (see Table 4).

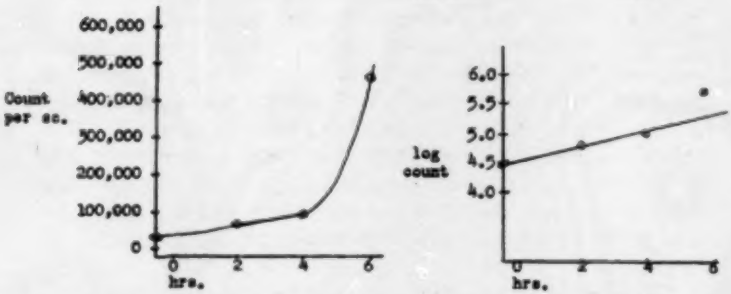
Table 4.

Mixer	*Am't Drunk*	*Count*
Me	control	1,300 per cc.
Me	1 oz.	5,500
Me	4 oz.	20,000
L	control	3
L	1 oz.	310
L	4 oz.	30,000

In the second set of regurgitation studies, the baby was allowed to drink 4 ounces from a full bottle. To plot the bacterial growth, a sample was plated immediately, and again at 2 hours, 4 hours, and six hours after, leaving the bottle at room temperature. (see Table 5).

Table 5.

Plated (hrs. after drinking)	*Count*	*Log*
control — before drinking	8 per cc.	0.90
0 hrs. — i.e. immediately after drinking	28,000	4.48
2 hrs.	60,000	4.78
4	100,000	5.00
6	450,000	5.65



As can be seen, a sizeable amount of bacteria is regurgitated immediately. This count then increases in approximately a regular logarithmic progression.

Is modern refrigeration adequate to halt bacterial growth in the formula? One would assume so, as Table 1 shows that milk refrigerated for 48 hours at 10°C. (50°F.) still had very low bacterial counts. To further check on this point, a formula of which the baby had drunk was refrigerated, therefore, starting with an initial sizeable contamination. Table 6 shows the bacterial counts before refrigeration, and after refrigeration for 48 hours at 10°C. (50°F.).

Table 6.

Sample	*Count per cc.* *Before Refrig.*	*After Refrig. 48 hr.*
#1	48,000	9,700
#2	70,000	50,000

It can be seen that modern refrigeration halts bacterial multiplication very effectively.

Gram-stains of colonies isolated in the formula mixing and regurgitation studies revealed gram-positive and negative cocci, and gram-negative rods.

IV. DISCUSSION

The results of these studies will interest the pediatrician and the general practitioner. There is a tendency now-a-days to be more lenient in demanding aseptic technique of mothers while mixing formula. With an excellent city water supply and modern refrigeration methods, the sterilization procedures demanded a number of years ago have begun to seem obsolete to some. The City of New York provides its citizens with an extremely good water supply, containing a low count of coliform organisms after chlorination.[12] The temperatures of most of our modern refrigerators are cold enough to repress bacterial growth in milk. The bacterial counts in milk supplied to the consumer are low due to rigid control standards, refrigeration and care. But, the physician must never forget the human element in the mixing of all formulas, and this element is enough to contaminate a formula significantly.

This experiment was done with intelligent mothers who maintained clean households. They mixed formulas with sterile water and almost sterile milk powder. The fact still remains that one of the group consistently contaminated the formula she mixed.

One might ask what is the definition of significant contamination; how do you know the formula will adversely affect the infant? The answer, obviously, is that we do not know. It depends on the type of bacteria introduced in the contamination, whether pathogenic or not. However, it is safer to feed an infant an almost sterile formula, than to feed him a formula with an unknown amount of contamination. Furthermore, it is hard to draw conclusions from the regurgitation studies. Although the infant may significantly contaminate his formula by regurgitation while he drinks a part of it, it is undeniable that the contaminating bacteria come from his own natural flora. Are these bacteria then pathogenic to him if he re-ingests them at a later time? One would say no, probably not. We have shown above (Table 6) that immediate refrigeration will halt further bacterial growth; on the other hand, if the formula bottle is left at room temperature for a number of hours, and not refrigerated immediately, it has been shown that the bacteria increase greatly in numbers. Whether or not the increased number of bacteria would now be pathogenic to the infant is rather difficult to determine scientifically, but there is no question that the infant will be imbibing a greater number of bacteria from the milk than he originally regurgitated into it.

How the physician should advise his patient concerning formula preparation and sterilization is one which must be decided personally. Much may depend on the purity of the city water supply, and the availability of adequate refrigeration. Mild infant diarrhea is a difficult disease to measure; much of it never comes to the attention of the physician, nevertheless, it may give trouble to the parents.

The purpose of this paper has been to show that formula contamination can easily occur, even with relatively clean technique, and that the milk remaining in a baby bottle after part has been drunk contains large numbers of bacteria, and should be treated accordingly.

V. SUMMARY AND CONCLUSIONS

1. An attempt was made to culture infant formulas as prepared in the home, to determine the amount of contamination introduced in mixing the formula.

2. Formula was cultured after the infant had drunk half of an 8-ounce bottle to determine the amount of contamination from regurgitation while sucking.

3. The bacterial titre was traced as the contaminated bottle was left at room temperature.

4. Modern refrigeration is adequate to halt bacterial growth in infant formula.

BIBLIOGRAPHY

1. Smith, F. R.: Terminal Heating of Infant Formula. I. Bacteriological Investigation of Low-pressure Technique. J. Am. Dietet. A., 24:755-759, 1948.
2. Finley, R. D.: Terminal Heating of Infant Formula. II. Bacteriological Investigation of High Pressure Technique. J. Am. Dietet. A., 24:760-763, 1948.
3. Lowe, R. H.: Formula Room Experiment in Terminal Sterilization. Mod. Hosp., 69: Aug. 1947.
4. Heinemann, H. E. O., Director Res. Div., Pet Milk Company. Personal Communication of Unpublished Data.
5. Holle, Henry, Commissioner, Texas State Dept. of Health. Experiment on Home Sterilization. Unpublished Data.
6. Infant Care, U. S. Dept. of Health, Educ. and Welfare. Children's Bureau Publication No. 8, 1951.
7. Formula Making. Nutrition Leaflet 2-60M-701265 (51)., City of New York, Dept. of Health.
8. Your Baby's Formula and How to Prepare It. Pet Milk Company, 1951.
9. Filer, L. J. Jr., Med. Director, M & R Laboratories, (Similac). Personal communication.
10. Giblin, J. and VonPourtales, J. H.: Am. J. Dis. Child., 41:1100-1103, May 1931.
11. Standard Methods for the Examination of Dairy Products. Am. Pub. Health A., Inc. 10th Ed., 1953.
12. Coliform Organisms in Routine Sampling of Public Water Before and After Chlorination, 1948-1953. City of New York, Dept. of Health, Bureau of Sanitary Engineering.

INCIDENCE OF RETROLENTAL FIBROPLASIA IN ENGLAND AND WALES IN 1951. J. T. Boyd and K. M. Hirst. Brit. M. J., 2:83-85, July 9, 1955. Particulars were obtained from all local authorities in England and Wales of infants born in 1951 having a birth weight of 4 lb. 6 oz. (2,000 gm.) or less and surviving at least two months. The survey provided data on 6,926 infants. The condition of the eyes was ascertained and 127, or 1.83%, of the infants were found to have retrolental fibroplasia. Not a single case of retrolental fibroplasia was found among the 797 infants who were born at home and remained there. This absence of cases among the home infants supports the view that the risk of developing retrolental fibroplasia is closely associated with some factor connected with hospitalization. Applying the specific incidence rates by sex and birth weight observed in the total population of infants to the number of infants in each of the corresponding subgroups of the home population, it was found that as many as seven or eight cases would have occurred among the infants born at home and not transferred to hospital, if they had been subject to the same risks as the whole population. The incidence of retrolental fibroplasia was closely related to birth weight, declining steeply with increasing birth weight. Considerable regional variability was observed.—*J.A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

TYPHOID INFECTION CONVEYED BY A CONVALESCENT INFANT*

THOMAS S. SOUTHWORTH, M.D.
New York.

As the outcome of the general awakening the world over to the unnecessarily large mortality from typhoid fever, searching investigations have been made into the common and uncommon sources of typhoid infection. Water, milk and shellfish repeatedly have been proven to be carriers of the infection. Epidemics have been traced to improper disposal and disinfection of the excreta of patients during the active period of the disease. At present, the laity as well as the medical profession are fairly well informed of the dangers which exist and the preventive measures to be employed during the continuance of the febrile movement.

More recently, however, the finger of suspicion has pointed to the convalescent, and urgent warnings have been issued by several investigators that the convalescent may still harbor the bacilli in sufficient numbers to constitute a serious menace to the public health. Thus far, only occasional proof has been forthcoming of actual transference of the disease by such convalescents. The difficulties attendant upon the establishment of such proof are considerable, since it is necessary to eliminate beyond reasonable doubt other possible sources of infection. The exceptional circumstances in the following account of two house epidemics of typhoid fever, between which a convalescent infant seems to have been the connecting link will, therefore, I trust, prove to be worthy of record.

The two families concerned in this history will be known for differentiation as the M—— family and the C—— family. The

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M—— family lived in Blissville, Long Island City, a suburb of Brooklyn; the C—— family in New York City. Their homes, therefore, were in different cities, separated by a wide river, and having different sources of water supply. Typhoid fever had been somewhat prevalent in Blissville when, in October 1905, the first child in the M—— family was taken sick with that disease. On November 22, 1905, another child, an infant, Teddy M., aged fourteen months, became ill with fever, vomiting and intestinal symptoms, during the course of which he developed a pneumonia. His physician states that his temperature most of the time was about 100°F., but it rose, for a few days only, to 102°F. Typhoid was suspected, but several Widal tests gave negative results. He had recovered from his illness before January 8, 1906, when his mother and two sisters, having developed typhoid, he was sent with them to St. John's Hospital, Long Island City, where his mother subsequently died. Still another boy was sent to friends in Elizabeth, N. J., where he is said to have been sick later in the hospital with typhoid-pneumonia.

Teddy M., the infant with whom we are especially concerned, remained in St. John's Hospital four days, during which time the house physician states that he had no fever. He was then, on January 11, 1906, taken to the home of the C—— family in New York. This family consisted of father, mother and four children. They occupied three connecting rooms, the middle one of which was kitchen, dining-room and living-room combined. In this room Teddy M. spent his days. He was pale and sickly, and it is said that his stools were thin, loose and smelled "fiercely." His diapers were usually disposed of quickly in the daytime, but were occasionally left about at night. January 27th, sixteen days after his arrival in the C—— family, I was asked to see him and found a strikingly pale child, now sixteen months of age, evidently rachitic, with large, square head and well developed rosary. Four days previously a somewhat redundant prepuce had become red and irritated, and on this day it was edematous and the tip raw and bleeding. Temperature 103.5°F. There was some cough, and physical examination showed generalized râles, but no evident consolidation. Two days later, under treatment, the temperature was normal, crackling râles right chest posteriorly, edema of prepuce gone and lesions healing rapidly.

February 1, twenty days after he entered the C—— family, two children of this family, eight and thirteen years of age, began

simultaneously to have headache, general malaise and fever; temperatures rose in a few days to 103.5°F., spleens became palpable, tongues heavily coated, and roseola appeared upon the abdomen of one of them. On the tenth day of the disease, Widal was positive in both cases, and they were sent to Roosevelt Hospital.

February 14, a third child of the C—— family developed temperature with similar symptoms in all respects, and despite a negative Widal on the sixth day was sent to Roosevelt Hospital, where the diagnosis of typhoid was subsequently confirmed. On the day that this third child went to the hospital, the C—— family, now thoroughly alarmed, sent Teddy M. back to St. John's Hospital.

February 22, through the courtesy of the House Physician at St. John's Hospital, a specimen of Teddy M.'s blood was obtained and submitted to the Board of Health, who reported a positive Widal reaction, thus proving him to have had typhoid fever at some time in the past.

We have, then, in these histories two house epidemics of typhoid fever in different cities, the connecting link between which was apparently an infant of sixteen months of age, who, preceded and followed by definite cases of typhoid in his own family, passed through an illness with prolonged but relatively moderate temperature, with gastrointestinal symptoms and a complicating pneumonia, without positive Widal at the time, but after some weeks giving a positive reaction. This infant, after a period of normal temperature for about two weeks, during part of which he was under observation in a hospital, then entered another family and house in which there had been no typhoid, and during his stay there had only one acute indisposition of a few days' duration, with temperature falling to the normal in two days under local treatment of an edematous and excoriated prepuce. Three weeks after his arrival there, two children came down with typhoid and two weeks later a third child. Painstaking inquiry revealed no other source of infection. The evidence certainly seems to point strongly to the infant Teddy M. as the carrier of infection after his convalescence.

The rooms of the second famly have been described. The infant in question being unable to walk, usually sat in a high chair in the combination kitchen, dining-room and living-room. Every possible opportunity was presented for contamination of food. The loose stools required frequent changing of the diapers by a

busy woman, who had five children to cook and care for. The single faucet of cold water over a sink usually filled with soiled dishes and utensils offered the most available facility for perfunctory hand-washing. Observation of this class of people with large families leads to the conclusion that hand-washing after the changing of diapers is performed only in case the hands are visibly soiled. Hands moist with the urine of a wet diaper are wiped upon the dry one with which the former is to be replaced. This unappetizing picture of domestic life among the artisan class requires no apology for its presentation, since it emphasizes the ample opportunities for the infection of the other children — an infection which seems to have been simultaneous in two of the cases.

In view of the different ways of possible typhoid conveyance in this case, a short summary of recent literature may be admissible at this point. The isolation of typhoid bacilli from the stools even at the present day offers such difficulties as to have limited investigations in this field. Dr. W. H. Park states that the typhoid bacilli are usually eliminated by the feces, being derived from ulcerated portions of the intestines. Their growth within the intestinal contents is, with few exceptions, not extensive. Although usually dying out quickly in the stools, which adds to the difficulty of isolation, in certain stools they multiply freely, finding a suitable soil. V. Drigalski and H. Conradi, who devised one of the quicker methods of differentiating the typhoid from colon bacilli, found typhoid bacilli as a diagnostic aid in the stools of one-half the cases where a 1 to 10 Widal test was still negative. Hiss, while finding typhoid bacilli in the stools in 80 per cent of the cases examined during the febrile stage, stated that the convalescent cases which he examined gave uniformly negative results, and this rapid disappearance after convalescence was apparently confirmed by others. This, however, is by no means the invariable rule.

Since the establishment of the Royal Institutions for Bacteriological Research, which have taken up the study and control of typhoid in certain districts of Germany, evidence is rapidly appearing of the spread of typhoid by persons long convalescent, who are now technically spoken of as "bacillus carriers." These are defined as those whose excreta contain the bacilli after ten weeks from the beginning of the illness, or from the beginning of the last relapse. Lentz at Trier discovered twenty-two typhoid carriers, to seven of whom other cases, often multiple, were traced. A. Besserer and

J. Jaffe found typhoid bacilli in the stools of four persons who had had typhoid three months, six months, four years and seven years previously. Seige also, who states that "contact infection" from these cases is coming more and more into prominence, reports two especially interesting cases, in which mothers having had typhoid and living thereafter under circumstances which apparently precluded reinfection, infected their children two years and ten years later, the bacilli being isolated from the mothers' stools four years and ten years respectively after recovery from the disease. Furthermore, V. Drigalski and Conradi have demonstrated the bacilli in the stools of four persons who remained entirely well after exposure to typhoid. Such reports, which could be multiplied, suffice to encourage the belief that continued search and newer methods yet to be devised may show that the bacilli persist in the stools even less exceptionally than is now believed, their persistence being due either to chronic infection of the gall bladder or to intestinal conditions which produce stools offering a favorable medium for their growth.

With the urine it is different. The infectious character of many urines during convalescence from typhoid is well established. Many investigators have worked in this field. Thus far it would appear that typhoid bacilli may be found in the urine of about 21 per cent. of the cases (Richardson). They are usually associated with albumin and renal casts, although these may both be present in the urine without the bacilli. Both Richardson and J. Jacobi have found typhoid bacilli in the urine as early as the eighth day of the disease, although they usually appear in the later weeks. Petruschky found them for the first time in 1 case six days after complete defervescence. All agree that if they appear at all they persist in the majority of cases into convalescence. Richardson's conservative statement is that they may persist for weeks, occasionally for months, rarely for years. Dönitz reports the finding of bacilli in the urine of two women four and seven months after their illnesses; the latter of whom infected her husband. When present, the bacilli are usually in pure culture and enormous numbers. Petruschky, previously quoted, estimated them in one case to be 172,000,000 in 1 cc. It is stated that such urines are often pale, abundant, somewhat cloudy and with a tendency to form a film on the surface upon standing. These conditions should at least excite suspicion.

The feces and urine of convalescent typhoid patients are, how-

ever, not the only sources of danger. The persistence of bacilli in pus as long as one year is perhaps of lesser importance; but it is worthy of note in connection with the history of the case which I have reported, that the bacilli are present and may persist for considerable lengths of time in the sputum of patients having a complicating bronchitis or pneumonia. Seven weeks is the longest period recorded.

While it has thus been established that typhoid bacilli may persist in the stools, urine, and possibly the sputum of patients convalescent from typhoid for considerable periods, a general recognition of this fact has not as yet entered sufficiently into prophylactic practice. The convalescent typhoid patient, except from certain hospitals or from the care of the more enlightened practitioner, is allowed to go his way without any special precautions. Disinfection of stools or urine ceases with the departure of the trained nurse or the discharge of the patient from active treatment. Fortunately, the adult and the older child when convalescent pass their urine and stools in the closet, so that the danger of infection through manual contamination is largely eliminated, the patient himself being presumably immune to reinfection. With the infant it is different, since the diaper or vessel is handled and the infant cleansed by some attendant. In the absence of any appreciation of the dangers, this may readily afford opportunities for the infection of the attendant or of others whose food she may prepare.

Such a case as that which I have related and the exceptional circumstances which made it possible to fix upon the infant as the link between the two outbreaks may aid in throwing additional light upon the origin of some of the apparently sporadic cases. It also again directs attention to the mild course which the disease may run, particularly in young children, and the possibility that it may escape recognition, especially if the appearance of the Widal reaction be delayed or the attention be diverted by complications. Unfortunately, we can only conjecture whether the infection was conveyed through the more probable agency of a bacteria-laden urine, or whether the chronic intestinal disturbance of a noticeably rachitic infant with foul stools produced a favorable soil for the persistance of the bacillus either in numbers or virulence.

Thus far, no effective method of eliminating the typhoid bacilli from the intestinal tract seems to have been discovered.

DEPARTMENT OF ABSTRACTS

Conducted by
MICHAEL A. BRESCIA, M.D., NEW YORK

SOBEL, G. R.: ADENOTONSILLECTOMY IN THE ALLERGIC CHILD. (New York State Journal Medicine, 56:3172, Oct. 15, 1956).

There is often no need for adenotonsillectomy in the allergic patient after successful antiallergic and anti-infective therapy. If indications for adenotonsillectomy still exist after treatment, it should be performed during a nonpollinating period. The nasal smear for eosinophils is helpful in diagnosing allergic conditions. The presence or absence of tonsils or adenoids has no bearing on the basic allergic pattern. AUTHOR'S SUMMARY.

FRENKEL, E. P.; SHIVER, C. B., JR.; BERG, P. and CARIS, T. N.: MENINGOENCEPHALITIS IN INFECTIOUS MONONUCLEOSIS. REPORT OF A CASE TREATED WITH CORTISONE. (Journal American Medical Association, 162:885, Oct. 27, 1956).

In a case of infectious mononucleosis with associated meningoencephalitis, recovery occurred rapidly after the administration of cortisone. As far as can be determined by us, this is the first time the use of cortisone has been reported in meningoencephalitis due to infectious mononucleosis. AUTHORS' SUMMARY.

PICK, W.: PARINAUD'S OCULOGLANDULAR SYNDROME. (Journal of Pediatrics, 49:316, Sept. 1956).

A case of Parinaud's oculoglandular syndrome is described in a 7 year-old boy who presented himself as a differential diagnostic problem of a preauricular tumor. The syndrome consists of a granulomatous conjunctivitis with associated regional lymphadenopathy. If an unexplained preauricular mass is found, the upper lid should be everted and examined for the conjunctivitis of Parinaud. Treatment with achromycin by mouth and sulfathiazole ophthalmic ointment locally shortens the self-limited course of this syndrome. Studies necessary to demonstrate an etiological agent are reviewed and it is emphasized that laboratory confirmation is difficult even under the best circumstances. AUTHOR'S SUMMARY.

THOMAS, W. A.; LEE, K. T.; MCGAVRAN, M. H. and RABIN, E. R.: ENDOCARDIAL FIBROELASTOSIS IN INFANTS ASSOCIATED WITH THROMBOSIS AND CALCIFICATION OF ARTERIES AND MYOCARDIAL INFARCTS. (New England Journal Medicine, 255:464, Sept. 6, 1956).

A study of the etiology and pathogenesis of calcific arterial disease of infants with myocardial infarcts is presented based on two autopsied patients with this disease, two infants with the left coronary artery arising from the pulmonary artery, twenty-four infants with uncomplicated endocardial fibroelastosis, one infant with endocardial fibroelastosis complicated by mural thrombosis and myocardial infarcts without calcific arterial lesions and five rabbits with myocardial infarcts resulting from thromboembolism. Both infants with calcific arterial disease and myocardial infarcts were found to have, in addition, endocardial fibroelastosis (characterized by fibroelastic thickening of the endocardium of the left ventricle and cardiac hypertrophy). This association has not been described in previous reports. Since both diseases are uncommon, it is unlikely that they are associated merely by chance. The relation between the two diseases can be explained in several ways. The most likely possibility seems to be that endocardial fibroelastosis was the underlying process and that the arterial disease and myocardial infarction resulted from thromboembolism. Evidence is presented supporting this hypothesis, and alternative possibilities are discussed.

AUTHORS' SUMMARY.

BROWN, H. W.; CHAN, K. F. and YOLKEN, H.: EFFICACY OF PROMETHAZINE AND PYRATHIAZINE AGAINST ENTEROBIASIS. (Journal American Medical Association, 162:1049, Nov. 10, 1956).

Promethazine (Phenergan) hydrochloride administration at bedtime in a single dose of 100 mg. or 125 mg. to fifteen children, weighing 27 pounds to 52 pounds, who were harboring Enterobius vermicularis resulted in a cure of 33 per cent of the children. Extreme drowsiness was experienced by twelve of the children, and three of them became extremely restless. Only two of the ten children given 100 mg. to 125 mg. of pyrathiazine (Pyrrolazote) hydrochloride lost their infection. These two compounds are likewise ineffective against syphacia obvelata, the pinworm of mice, even when given in large doses.

AUTHORS' SUMMARY.

Paternalism and Mental Health. (Northwest Medicine, 55:763, July 1956).

Those who deplore paternalism in government will find support for their views in a recent comment about its effect on mental health. The comment was made in Lewiston by John R. Butler, Director of the Division of Mental Health of Idaho. While in Europe, he observed the results when too much social responsibility is assumed by government. His program in Idaho is based on local activity to meet local needs. His insistence on utilization of local resources is due to the conditions he encountered in Europe. Dr. Butler stated that he was appalled by conditions of mental health in Europe, particularly in Sweden. As welfare responsibilities are taken over by government, he finds that individual and family responsibilities lessen. As this occurs, mental health deteriorates. He pointed out that in our own country when there is a lessening of family responsibility, there is demand for the community to step in. If the community does not provide satisfaction, the state is asked for assistance for the problems of its citizens. Mental health suffers when this happens. The process is seen with remarkable clarity in Sweden. There the government has assumed a high level of social responsibility. The welfare of citizens is assured by the state. "Yet," Dr. Butler says, "Sweden has the highest incidence of mental disease, the highest youth delinquency, the highest rate of alcoholism and the highest percentage of illegitimate children of any nation in the western world. I was amazed at the number of purposeless individuals — many of them alcoholics — walking the streets when I visited there." EDITORIAL.

ANDERSON, O. W.: TREATMENT OF LABIAL ADHESIONS IN CHILDREN. (Journal American Medical Association, 162:951, Nov. 3, 1956).

Labial adhesions are common in certain children, possibly due to some peculiar tenaciousness of the mucus secretions on the surfaces of the labia. Every doctor should recognize these adhesions in order to avoid causing alarm to parents. These adhesions seldom cause any trouble, but, if symptoms should develop which can be attributed to the presence of the adhesions, then the labia should be separated. These adhesions cause little trouble; their removal is painful, and since they tend to recur and do tend to disappear with maturity, a "no treatment plan" should be tried.

AUTHOR'S SUMMARY

WEST, M. and ZIMMERMAN, H. J.: HEMOLYTIC ANEMIA IN PATIENT RECEIVING NITROFURANTOIN (FURADANTIN). (Journal American Medical Association, 162:637, Oct. 13, 1956).

A case of hemolytic anemia occurred in a patient receiving nitrofurantoin. No other known factor could be established as being responsible for this phenomenon. The patient had never had any similar episodes in the past. Hemolysis subsided after the administration of the drug was discontinued, and has not recurred during a 6 month period of observation. It seemed reasonable to conclude that the hemolytic anemia was related to the administration of nitrofurantoin, although this was not proved unequivocally.

AUTHORS' SUMMARY.

Vaccination against Whooping-Cough. Relation between Protection in Children and Results of Laboratory Tests. A Report to the Whooping-Cough Immunization Committee of the Medical Research Council and to the Medical Officers of Health for Cardiff, Leeds, Leyton, Manchester, Middlesex, Oxford, Poole, Tottenham, Walthamstow, and Wembley. (British Medical Journal, 4990:454, Aug. 25, 1956).

In two series of field trials, 14 pertussis vaccines were tested for their protective potency in 28,799 children. The vaccines used in the first series of trials were poor, but those in the second series gave substantial protection, which was maintained for at least two and a half to three years after vaccination. At the same time as the field trials were in progress, laboratory tests were made in which the vaccines were tested for their ability to protect mice against intracerebral pertussis infection. Some of the vaccines were tested also for their ability to produce specific agglutinins in mice and in children. A comparison between the field and laboratory results showed some correlation between the potency of vaccines in protecting children and their ability to protect mice against intracerebral infection. There was also evidence of a correlation between protection in children and ability to produce agglutinin in both mice and children. It is concluded that in future only those pertussis vaccines which have been shown, by the intracerebral mouse-protection test, to have an adequate potency in relation to the British standard pertussis vaccine should be issued for use in children.

AUTHORS' SUMMARY.

BOOK REVIEWS

Conducted by
MICHAEL A. BRESCIA, M.D., NEW YORK

THE MENTALLY RETARDED PATIENT. By Harold Michal-Smith, Ph.D. Cloth. Pp. 203. Price $4.00. Philadelphia, Pa.: J. B. Lippincott Co., 1956.

There is a great need for books on mental retardation, if for no other reason than to emphasize the magnitude and importance of the problem. The present volume, although interesting and comparatively easy reading deals mainly in generalities. Progress can be made in this most difficult field if some specific etiological factor or factors could be found which might be subject to specific modalities of treatment or prevention. In attempting to explain brain impairment, the author states, not too clearly, viz.: "The brain-damaged patient is suffering from a loss rather than a lack. He has blocked neurologic connections, not missing connections. A damaged rather than a basically defective mechanism is the problem." This reviewer feels that the use of the term "brain-damaged" in reference to mental retardation, since it commonly connotes physical violence as a definite and common cause of mental retardation, is ill advised. The author himself agrees with this opinion in stating: "The idea that accidents and birth injuries are always to blame for mental retardation can make scapegoats of doctors and labor-room personnel. Actually, in a relatively small number of cases, brain-damage can be seen directly traceable to a blow on the head, a known birth injury or mishap during pregnancy." This book can be profitable to the reader. One can obtain an inkling of the complexity of the problem and the multiplicity of etiological factors involved from the chapter on Classification of Mental Retardation by Dr. Lawrence B. Slobody. M.A.B.

ANGIOCARDIOGRAPHIC INTERPRETATION IN CONGENITAL HEART DISEASE. By Herbert L. Abrams, M.D. and Henry S. Kaplan, M.D. Cloth. Pp. 233. Illustrated. Price $11.75. Springfield, Ill.: Charles C. Thomas, 1956.

This is an excellent and well done volume demonstrating by numerous photographs the various abnormal configurations of the

heart as delineated by contrast media. The schematic drawings are a great aid in interpreting the x-rays. Practically all the important congenital malformations of heart and blood vessels are demonstrated. To those who are interested in cardiography, the book is of great value, but it has only limited value to the pediatrician except for occasional reference. M.A.B.

CHILD BEHAVIOR. By Frances L. Ilg, M.D. and Louise Bates Ames, Ph.D. Cloth, Pp. 364. Price $3.95. New York: Harper & Brothers, 1956.

This is a most useful book for the mother who needs help in rearing her children, and who is able to apply and appreciate the written word. The book discusses, most succinctly, the most common and ubiquitous problems such as sleep, eating, elimination, etc. In this regard, the book becomes an aid to the pediatrician in that it can complement and supplement his explanations to the mother. I certainly agree with Gesell's foreword to the volume, viz: "The topics are discussed concretely, but not too dogmatically. The approach is straightforward, the style direct." The book can be recommended by the pediatrician to the mother whom he feels needs additional help, and both will profit thereby. M.A.B.

PRACTICAL PEDIATRIC DERMATOLOGY. By Morris Leider, M.D. Cloth. Pp. 433. Illustrated. Price $10.50. St. Louis: The C. V. Mosby Company, 1956.

This is a very commendable book particularly because it does not belie the title by being a most practical book. This book will appeal to the pediatrician not only because the text is concise and geared particularly to the non-dermatologist, but also because of the many fine photographs of the various skin conditions that one might encounter. The pictures are large, clear and demonstrate the skin lesions well. A special section of blue paper for easy reference contains 101 useful dermatologic preparations. This section in itself greatly enhances the value of this book. A glossary of words at the end of the book reveals that the author has maintained his sense of humor in writing this book, and the reader might regain some of his own in reading some of the author's personal comments. This book is highly recommended. M.A.B.

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